RADON
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CARCINOGENICITY

Radon and its isotopic forms, radon-222 and radon-220 are *known to be human carcinogens* based on sufficient evidence of carcinogenicity in humans (IARC V.43, 1988; ATSDR, 1990-K025). Increased incidences of lung cancer have been reported from numerous epidemiologic studies of groups occupationally exposed to high doses of radon, especially underground hard rock miners. These include particularly uranium miners, but also groups of iron-ore and other metal miners, and one group of fluorspar miners. Strong evidence for exposure response relationships has been obtained from several studies, in spite of uncertainties that affect estimates of the exposure of the study populations to radon decay products. Several small case-control studies of lung cancer have suggested a higher risk among individuals living in houses known or presumed to have higher levels of radon and its decay products than among individuals with lower presumed exposure in houses. The evidence on the interaction of radon and its decay products with cigarette smoking with regard to lung cancer does not lead to a simple conclusion. The data from the largest study are consistent with a multiplicative or submultiplicative model of synergisms and reject an additive model. In many studies of miners and in one of presumed domestic exposure, small cell cancers accounted for a greater proportion than expected of the lung cancer cases. In one population of uranium miners, this proportion has been declining with the passage of time. Because of the limited scale of epidemiologic studies of nonoccupational exposure to radon decay products available at the time reviews were made, quantification of risk has been based only on data of miners' experience. An IARC Working Group considered that the epidemiologic evidence does not lead to a firm conclusion concerning the interaction between exposure to radon decay products and tobacco smoking. Most of the epidemiologic studies involve small numbers of cases, and the analytical approaches for assessing interaction have been variable and sometimes inadequate.

An IARC Working Group reported that there is sufficient evidence of carcinogenicity of radon in experimental animals (IARC V.43, 1988; ATSDR, 1990-K025). When administered by inhalation, preceded by a single exposure to cerium hydroxide dust, radon induced pulmonary adenomas, adenocarcinomas, invasive mixed adenosquamous carcinomas, and squamous cell carcinomas in male rats. Extrapulmonary metastases occurred in only one animal. Most or all of the tumors were believed to be bronchiolar or bronchio-alveolar in origin. Radon decay products in combination with uranium-ore dust induced a progression of activity from single basal cell hyperplasia in bronchioles to malignant tumors in male hamsters when exposed by inhalation. Lung tumors observed were adenomas, adenocarcinomas, and squamous cell carcinomas; bronchiolar and alveolar metaplasia, adenomatous lesions, fibrosis, and interstitial pneumonia were also observed. When administered by inhalation in combination with decay products, uranium-ore dust, and cigarette smoke, radon-induced nasal carcinomas, epidermoid carcinomas, bronchio-alveolar carcinomas, and fibrosarcoma were observed in dogs of both sexes. In general, a significant increase was observed in respiratory tract tumors in rats and dogs in comparison with unexposed animals. A dose-response relationship was noted in those experiments with rats in which radon was tested. In most instances, tumors at sites other than the lung were not reported, but in one study, mention was made of tumors of the upper lip and urinary tract in rats.

PROPERTIES

Radon is a colorless, odorless gas that is nine times denser than air. It is also fairly soluble in water and organic solvents. Although reaction with other compounds is comparatively rare, it is not completely inert and forms stable molecules with highly electronegative materials. Radon is considered a noble gas that occurs in several isotopic forms. Only two are found in significant concentrations in the human environment: radon-222 and radon-220. Radon-222 is a member of the radioactive decay chain of uranium-238, and radon-220 is formed in the decay chain of thorium-232. Radon-222 decays in a sequence of radionuclides called radon decay products, radon daughters, or radon progeny. It is radon-222 that most readily occurs in the environment. Atmospheric releases of radon-222 results in the formation of decay products that are radioisotopes of heavy metals (polonium, lead, bismuth) and rapidly attach to other airborne materials (IARC V.43, 1988) such as dust and other materials facilitating inhalation.

USE

Radon is a noble gas. Only two of its isotopic forms are found in significant concentrations in the human environment: radon-222 and radon-220. Their decay products are not gases and occur as unattached ions or atoms, condensation nuclei, or attached to particles. This decay product of uranium-238 is commonly found in uranium mines. Radon has been used in some spas for presumed medical effects (IARC V.43, 1988).

In addition, radon is used to initiate and influence chemical reactions and as a surface label in the study of surface reactions. It has been obtained by pumping the gases off of a solution of a radium salt, sparking the gas mixture to combine the hydrogen and oxygen, removing the water and carbon dioxide by adsorption, and freezing out the radon (Merck, 1989). It is also used in cancer treatment, as a tracer in leak detection, and radiography (HSDB, 1997).

PRODUCTION

Radon is a naturally occurring radioactive gas and comes from the natural breakdown (radioactive decay) of uranium. Most soils contain varying amounts of uranium. It is usually found in igneous rock and soil, but in some cases, well water may also be a source of radon. Radon was produced commercially for use in radiation therapy but for the most part has been replaced by radionuclides made in accelerators and nuclear reactors. Radon production can be found in research laboratories and universities for use in experimental studies (ATSDR, 1990-K025; HSDB, 1997).

EXPOSURE

The primary routes of potential human exposure to radon are inhalation and ingestion. Radon in the ground, water supply, or building materials enters working and living spaces and disintegrates into its decay products. In comparison with levels in outdoor air, the concentrations of radon and its decay products to which humans are exposed in confined air spaces, particularly in underground work areas such as mines and buildings, are elevated. Although high concentrations of radon in groundwater may contribute to human exposure through ingestion, the radiation dose to the body due to inhalation of radon released from water is usually more important. This is because radon has a short residence time in the stomach, therefore contributing a small dose to the stomach (IARC V.43, 1988; ATSDR, 1990-K025).

Concentrations of radon decay products measured in the air of underground mines throughout the world vary by several orders of magnitude. In countries for which data were available, concentrations of radon decay products in underground mines are now typically less than 1000 Bq/m^3. The average radon concentrations in houses are generally much lower than the average radon concentrations in underground ore mines. Workers are exposed to radon in several occupations. Underground uranium miners are exposed to the highest levels of radon and its decay products. Other underground workers and certain mineral processing workers may also be exposed to significant levels. Emanation of radon from ordinary rock and soils and from radon-rich water can cause significant radon concentrations in tunnels, power stations, caves, public baths, and spas. In comparison to the average values for the continental United States (4.4 to 11 Bq/m^3), the highest concentrations of radon were found in the Colorado Plateau (18.5 to 30 Bq/m^3). Peripheral lymphocyte chromosomes from 80 underground uranium miners and 20 male controls in the Colorado Plateau were studied. Taken into account were confounding factors such as cigarette smoking and diagnostic radiation. Groups that were increasingly exposed to radon and its decay products were selected. Significantly more chromosomal aberrations were observed among miners with atypical bronchial cell cytology, suspected carcinoma, or carcinoma in situ than among miners with regular or mildly atypical cells, as evaluated by sputum cell cytology (IARC V.43, 1988; ATSDR, 1990-K025).

The Environmental Protection Agency (EPA) and the Surgeons General's Office have urged widespread testing for radon. They estimated that as many as 20,000 lung cancer deaths are caused each year by radon. Next to smoking, radon is the second leading cause of lung cancer. EPA says that nearly 1 in 3 homes checked in seven states and on three Indian lands had screening levels over 4 pCi/L (Chem. Week, 1988), the EPA's recommended action level for radon exposure.

Radon is a national environmental health problem. Elevated radon levels have been discovered in virtually every state. The EPA estimates that as many as 8 million homes throughout the country have elevated levels of radon. State surveys to date show that 1 out of 5 homes has elevated radon levels. Radon seeps into homes from the surrounding soil through cracks and other openings in the foundation. Indoor radon has been judged to be the most serious environmental carcinogen to which the general public is exposed and which the EPA must address. Based on current exposure and risk estimates, radon exposure in single-family houses may be a causal factor in as many as 20,000 of the total lung cancer fatalities which occur each year. Radon decay products (polonium-218 and polonium-214, solid form) can attach to the surface of aerosols, dusts, and smoke particles which may be inhaled, and become deeply lodged or trapped in the lungs. Once lodged, they can irradiate and penetrate the cells of mucous membranes, bronchi, and other pulmonary tissues.

Some scientific studies of radon exposure indicate that children may be more sensitive to radon. This may be due to their higher respiration rate and their rapidly dividing cells, which may be more vulnerable to radiation damage.

REGULATIONS

EPA regulates radon under the Clean Air Act (CAA), Clean Water Act (CWA), Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA), Resources Conservation and Recovery Act (RCRA), Safe Drinking Water Act (SDWA), Toxic Substances and Control Act (TSCA), and Superfund Amendments and Reauthorization Act (SARA). Congress has recognized the health consequence of radon and passed legislation in October 1988 that establishes a national goal which states that indoor radon levels should not

exceed ambient outdoor radon levels of 0.2 to 0.7 pCi/L. This law also recognizes the need for radon testing in schools and workplaces, and provides funds for states to initiate a variety of radon activities. Radon is a hazardous pollutant of air and water. EPA has established water quality criteria for radon, effluent guidelines, rules for general threshold amounts, and requirements for handling and disposal of radon containing wastes. In general, radon-220 and radon-222 emissions are limited to 20 pCi/m^2-s into the air. A reportable quantity (RQ) of 0.1 Ci has been established for radon-220 or radon-222 under CERCLA. NIOSH recommends that the exposure limit not exceed 1 working level month (WLM) per year and 0.083 working level (WL) per shift. OSHA regulates radon under the Hazard Communication Standard and as a chemical hazard in laboratories. Regulations are summarized in Volume II, Table A-32.